imaging-based suspect diagnosis, as highlighted in the editorial by Garcia-Pavia and de la Pompa (5) with a review of all the imaging criteria available to the date.

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REPLY: The Importance of Cardiac Cycle in the Imaging Criteria for Left Ventricular Noncompaction

We appreciate the letter by Dr. Vidal-Perez and colleagues as it highlights the current lack of consensus on nosology and diagnostic criteria of left ventricular noncompaction (LVNC). In daily clinical practice, the presence of noncompaction in the absence of dilated cardiomyopathy (DCM) or left ventricular hypertrophy (LVH) is often annotated as a descriptive finding but not systematically labeled as cardiomyopathy, especially when identified as an incidental finding in otherwise functionally normal hearts. In clinical reports, the term *hypertrabeculation* or *increased trabeculation* followed by topographic indications is far more common than the diagnosis of LVNC. The question remains as to when should LVNC also be diagnosed as LVNC cardiomyopathy (1)?

Imaging provides quantification on the anatomy of the layers of the left ventricle as well as the possibility of measuring ratios between compacted and noncompacted layers. The thickness of the left ventricular layers are taken in end-diastole with cardiac magnetic resonance (2). However, thickness of the left ventricular layers can be measured with echocardiography in both end-systole and end-diastole, as indicated by Jenni et al. (3) and Paterick et al. (4). Paterick et al. (4) selected to measure in end-diastole because the thickness of compacted and noncompacted layers was deemed more precise in end-diastole. Chamber wall thickness measurements performed with echocardiography in end-diastole is currently consistent with the convention of the American Society of Echocardiography. The absence of consensus criteria is partly explained by the still limited knowledge on the natural history of the disease, namely, the evolution of LVNC with normal left ventricular size and function at onset and later developed left ventricular dilation and eventually dysfunction or LVH.

The point raised by Vidal-Perez and colleagues about genetics is appropriate more as a question rather than an answer as genes only causing LVNC and not associated with DCM or hypertrophic cardiomyopathy have not been identified. Although we strongly support genetic testing in all cardiomyopathies, a key problem remains the definition of LVNC and consequently its diagnosis and distinction with features of prominent, increased trabeculation. The measurement of the thickness of the epicardial compacted layer could add information to the ratio of compaction/noncompaction. For example, a thick epicardial layer could guarantee a normal function whatever the noncompaction is.

A *major* clinical problem specifically associated with LVNC is the risk of mural endocardial thrombotic stratification and thromboembolic complications. The presence of deep recesses may favor the formation of mural thrombi, but this only occurs in dysfunctional hearts. No case to date with LVNC and normal cardiac function has been associated with mural thrombosis or embolic complications. These latter cases occur typically in dilated and dysfunctional hearts with LVNC, which is DCM with LVNC. The open question is when to start anticoagulant treatment in these patients.

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